summaries, which are given to the patients on discharge. But no efforts have been made to provide a monitoring service for the patients after discharge. They are given a supply of eyedrops and other medication as necessary and the summary in their native tongue, with instructions to go to their general practitioner should they experience difficulties. This lack of proper follow up is unacceptable to the ophthalmic profession in Britain and, I imagine, the other countries from which these patients originate.

I have offered to try to set up a postoperative service for British patients and should be pleased to hear from fellow ophthalmologists who are prepared to provide this service. It is no small problem: in the 18 months that the eye clinic has been in Gibraltar over 3000 British patients have been treated. From conversations I had with compatriots on board, the long waiting lists for NHS consultations and operations were a major factor in their decision to go to Gibraltar.

D P CHOYCE
Honorary consulting ophthalmic surgeon
London W1M 7DG

1 Tremlett G. Ophthalmologists protest at floating eye clinic. *BMJ* 1994;309:359. (6 August.)
2 Choyce DP. Floating eye clinic. *BMJ* 1994;309:1021. (15 October.)

Histopathology and medical laboratory scientific officers

EDITOR,—Arguing about whether pathologists or technicians should dissect gross specimens and select histopathological blocks overlooks a more fundamental requirement.[1,2] Qualifications apart, it is important that the same person handles both the gross and the microscopic examination of each specimen; the two steps ought never to be dissociated. To separate them not only damages the quality of the reporting but, if done regularly, retards the educational development of the pathologist.

The aim of gross examination is often misunderstood. It is not merely to record striking, measurable, and undeniable features with which to rebut future charges of misidentification. Nor is it to construct fluent strings of descriptive clichés, of questionable circumstantial accuracy, for the edification of the impressionable. The paramount aim is to apply experienced, skilled observation to the selective removal of those parts likely to yield microscopically diagnostic features.

The ability to recognise fruitful sites for block taking in large and complex specimens comes, if slowly, from the cyclical, iterative, process of regularly correlating the recollected naked eye appearances with the corresponding microscopic ones. Those whose gross dissections are not closely and repeatedly linked to the microscopic appearances may well succeed in capturing fairly obvious lesions, but lesions of any subtlety will escape them, resulting in diagnostic nullity, compromise, or inconclusion.

Learning, maintaining, and regularly extending the art of correlation take time and effort beyond those needed for diagnosis alone. They demand self discipline and diligence because, when diagnostic demands are heavy, their cultivation may be sacrificed to expediency. Correlation entails a progressive retreat from the higher magnifications needed to confirm diagnostic suspicions. As the powers are reduced more extensive, but less detailed, vistas are appreciated and retained. Examination of the section with a hand lens and the naked eye completes the mental matching to the site whence it came. Thus do the delicate alterations of texture, colour, translucency, and expansion in the gross specimen acquire microscopic reality. Success demands regular, conscientious, repetition.

The Royal College of Pathologists should insist on the continuity of pathological care by endorsing the intrinsic unity of the complementary gross and microscopical examinations. Without the college's support British pathological reporting and continuing education will sustain irreparable damage in the present managerially controlled decline.

STEWART FLETCHER
Consultant pathologist
Al-Zahra Hospital,
PO Box 3499,
Sharjah,
United Arab Emirates

1 Ashworth TG. The future of histopathology: protectionism or prudence? *BMJ* 1994;309:417. (6 August.)
2 Histology and medical laboratory scientific officers [letters]. *BMJ* 1994;309:803-5. (24 September.)

Reiter's syndrome attributed to hepatitis B immunisation

EDITOR,—Wajahat Hassan and Roger Oldham describe a case of Reiter's syndrome, which they attribute to immunisation against hepatitis B, which has not been reported previously.[1] We fail to understand how they can claim this when they did not exclude one of the commonest triggers for Reiter's syndrome—namely infection with *Chlamydia trachomatis*.[2] No urethral swab was taken for culture of chlamydia or detection of antigens, and no mention is made of serological testing for antibodies to *C trachomatis*, which are often significantly increased in Reiter's syndrome associated with chlamydia.[3] Although the patient had "no prodromal symptoms," it is well known that genital infections with *C trachomatis* may be asymptomatic. This omission is important for two reasons: firstly, evidence exists that a prolonged course of tetracycline may shorten the duration of arthritis if *C trachomatis* is the cause[4]; and, secondly, a diagnosis of chlamydial infection has important implications for any sexual partners since *C trachomatis* is the commonest cause of pelvic inflammatory disease with its sequelae of chronic pelvic pain, tubal pregnancy, and infertility.

Patients presenting with Reiter's syndrome without a history of an enteric pathogen as a trigger should be referred for a full screen for sexually transmitted infections.

P A FRASER
Senior registrar
J D WILSON
Consultant
Department of Genitourinary Medicine,
General Infirmary at Leeds,
Leeds LS1 3EX

1 Hassan W, Oldham R. Reiter's syndrome and reactive arthritis in health care workers after vaccination. *BMJ* 1994;309:94. (9 July.)
2 Keat A. Reiter's syndrome and reactive arthritis in perspective. *N Engl J Med* 1983;309:1606-15.
3 Martin DH, Pollock S, Kuo C, Wang S, Brunham RC, Holmes KK. Chlamydia trachomatis infections in men with Reiter's syndrome. *Ann Intern Med* 1984;100:207-13.
4 Lauhia A, Leirisalo-Repo M, Lähdevirta J, Saikku P, Repo H. Double-blind, placebo-controlled study of three-month treatment with lymecycline in reactive arthritis, with special reference to chlamydia arthritis. *Arthritis Rheum* 1991;34:6-14.

Hyperbaric oxygen treatment for crush injury

EDITOR,—Andrew D Shaw and colleagues draw attention to the self perpetuating process of oedema and ischaemia after crush injury and the need to break this cycle.[1] In ischaemia, failure of oxygen delivery is at the root of the problem. The cycle can be interrupted by giving oxygen at a high dose, provided that it is used early. In the United States crush injury is a recognised indication for hyperbaric oxygen treatment, which is paid for by insurers when given for this indication. This was discussed in an editorial in 1993.[2]

Although it has been hypothesised that additional oxygen may worsen reperfusion injury, the reverse has proved to be the case. In an experimental study Nylander *et al* found that a single 45 minute session of hyperbaric oxygen significantly reduced postischaemic oedema and that the effect lasted 48 hours.[3] Zamboni *et al* found that hyperbaric oxygen abolished neutrophil adhesion and minimised oedema after four hours of total ischaemia in rat gracilis muscle.[4] They are now using hyperbaric oxygen routinely in the reimplantation of extremities and report complete muscle survival and minimal soft tissue oedema with ischaemia times of up to 12 hours. Crush injury is often accompanied by hypotension and fat embolism causing cerebral oedema, which can also benefit from hyperbaric oxygen treatment.[5]

P B JAMES
Senior lecturer in occupational medicine
Wolfson Hyperbaric Medicine Unit,
University of Dundee,
Ninewells Hospital and Medical School,
Dundee DD1 9SY

1 Shaw AD, Sjølin SV, McQueen MM. Crush syndrome following unconsciousness: need for urgent orthopaedic referral. *BMJ* 1994;309:857-9. (1 October.)
2 Kindwall EP. Hyperbaric oxygen. *BMJ* 1993;307:515-6.
3 Nylander G, Lewis D, Nordström H, Larsson J. Reduction of postischemic edema with hyperbaric oxygen. *Plast Reconstr Surg* 1985;76:596-600.
4 Zamboni WA, Roth AC, Russell RC, Graham B, Suchy H, Kucan JO. Morphological analysis of the microcirculation during reperfusion of ischaemic skeletal muscle and the effect of hyperbaric oxygen. *Plast Reconstr Surg* 1993;91:1110-21.
5 Sukoff MH, Ragatz RE. Hyperbaric oxygenation for the treatment of acute cerebral edema. *Neurosurgery* 1982;10:29-38.

Skin necrosis induced by streptokinase

EDITOR,—J Penswick and A L Wright report a case of skin necrosis possibly induced by treatment with streptokinase.[1] Pathophysiologically they suspected "a variety of disturbances of the clotting and thrombolytic cascade," but they fail to give any evidence for their suspicion. The clinical course of events is compatible with cholesterol crystal embolisation. Cholesterol crystal embolisation of the arteries of the skin may present as cutaneous purpura and ulcers. It occurs in patients with extensive atherosclerotic disease as a consequence of the showering of cholesterol rich material from ulcerated atheromatous plaques into the arterial circulation. Cholesterol embolisation has been described after angiographic procedures, vascular surgery, and anticoagulant treatment and may manifest itself in multiple organ systems.[2]

Several case reports have described cholesterol crystal embolisation temporally and possibly causally related to treatment with streptokinase as well as to treatment with tissue plasminogen activator.[3] The time interval between the start of lytic treatment and the onset of symptoms of cholesterol crystal embolisation varied between seven hours[4] and four days.[5] The symptoms included livedo reticularis, myalgias, necrosis of the skin and toes, ulcers, gangrene of the hands and feet, and renal failure. Two cases of suspected cholesterol crystal embolisation after the application of streptokinase (Streptase) have been reported to Behringwerke AG between 1980 and 1994 as part of postmarketing surveillance. The interval between application and the onset of symptoms was 10 hours in one case and three days in the other.

On the basis of the patient's history (hypertension, peripheral vascular disease), the interval between the start of lytic treatment and onset of symptoms (24 hours), and the symptoms (diffuse tenderness, bruising of buttocks and upper thighs,